# A database of fibrillogenic mutations and experimental conditions.

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Amyloidoses such as Alzheimer's disease are degenerative disorders characterised by the presence of pathogenic fibrillar deposits throughout the body. Over twenty proteins have been identified as amyloidogenic, and despite there being little or no sequence, or fold, homology between them, the fibrils share similar morphological and tinctorial properties.

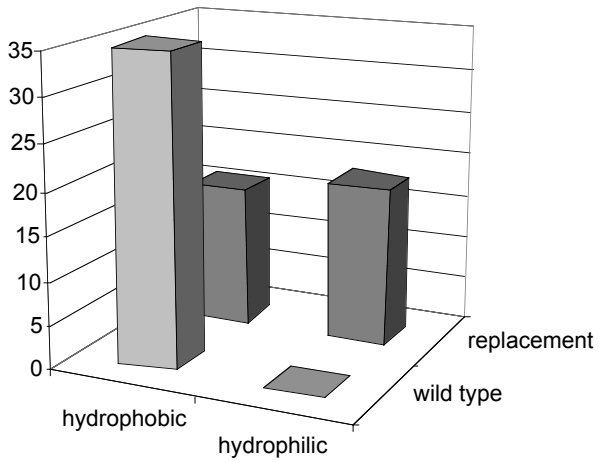


**Figure**. Hydrophobicity of replacement residues in mutations promoting amyloid fibril formation where the wild-type residue is hydrophobic and located in the protein structural core, illustrating a tendency for replacement with destabilizing hydrophilic residues.

We have constructed the Fibril_one database, which contains mutations and experimental conditions associated with fibril formation. There is an online user interface to the database enabling the user to identify trends associated with fibrillogenesis and specific mutation details associated with the amyloidgenic proteins. Trends identified in the database, suggest mutations promoting fibril formation have a strong association with destabilisation of the native fold.

Future work will focus primarily on the edge strands of beta proteins which are free to interact with other edge strands that they encounter. Natural beta proteins seem to be designed to specifically avoid this problem, however specific mutations may disrupt these protective mechanisms resulting in protein aggregation. Using machine learning methods can we predict and understand the property of edge strands from the protein sequence. Following this is it possible to predict mutations that are likely to lead to protein aggregation into the fibrillar structure, enhancing our understanding of the fibrillogenic mechanism in general.

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